

VINYL CHLORIDE

Identified as a toxic air contaminant under California's air toxics program (AB 1807) in 1990.

CAS Registry Number: 75-01-4

$\text{H}_2\text{C}=\text{CHCl}$

Molecular Formula: $\text{C}_2\text{H}_3\text{Cl}$

Vinyl chloride monomer is a sweet smelling, colorless gas at ambient temperature and pressure, and polymerizes in light or in the presence of a catalyst. Vinyl chloride is readily flammable and forms explosive mixtures in air. Upon combustion, it is degraded mainly to hydrogen chloride gas, carbon monoxide, carbon dioxide, and traces of phosgene. Vinyl chloride is expected to volatilize rapidly from water systems. It is soluble in ethanol, industrial solvents and some organic liquids but only slightly soluble in water (Merck, 1989). Studies have determined that distilled water spiked with 16 parts per million (ppm) vinyl chloride lost 96 percent of the vinyl chloride within 2 hours (U.S. EPA, 1974).

Physical Properties of Vinyl Chloride

Synonyms: chloroethene; chloroethylene; chlorethene; ethylene monochloride; Trovidur

Molecular Weight:	62.5
Boiling Point:	-13.37 °C at 760 mm Hg
Melting Point:	-153.8 °C
Flash Point:	-77.8 °C (open cup)
Vapor Pressure:	2530 mm Hg at 20 °C
Vapor Density:	2.15 (air = 1)
Density/Specific Gravity:	0.912 at 20/4 °C
Henry's Law Constant:	0.0560 atm/cv m-mole
Log Octanol/Water Partition Coefficient:	1.36
Conversion Factor:	1 ppm = 2.56 mg/m ³

(HSDB, 1995; Merck, 1989; Sax, 1989; U.S. EPA, 1994a)

SOURCES AND EMISSIONS

A. Sources

Vinyl chloride is used in the production and fabrication of polyvinyl chloride, ethylene dichloride, methyl chloroform, caprolactam, vinyl acetate, and vinylidene chloride (HSDB, 1995). Polyvinyl chloride (PVC) can be fabricated into several products such as PVC pipes, pipe fittings, and plastics. Residual vinyl chloride (RVC) in PVC ranges from

0.002 to 10 parts per million by weight (U.S. EPA, 1982b). When PVC is fabricated into final products, vinyl chloride is emitted (ARB, 1990d).

In general, the primary source of vinyl chloride is from landfills directly from disposed wastes which contain vinyl chloride and indirectly from the formation of vinyl chloride from the biodegradation of chlorinated hydrocarbons. Other minor mechanisms by which vinyl chloride emissions may occur include chemical reactions such as pyrolysis, surface photolysis, and hydrolysis of trichloroethylene and other chlorinated hydrocarbons, and off-gassing of PVC (Moulton et al., 1987). Publicly-owned treatment works (POTWs) treat wastewater which can contain vinyl chloride and halogenated compounds from industries. Vinyl chloride can be volatilized during treatment processes and chlorinated hydrocarbons such as trichloroethylene and 1,2-dichloroethane can be biodegraded to vinyl chloride (ARB, 1990d).

Landfills, POTWs, and PVC production are the major identified sources of vinyl chloride emissions in California. A variety of steps have been taken to reduce vinyl chloride emissions. A Suggested Control Measure for Landfill Gas Emissions, prepared by the California Air Pollution Control Officers Association (CAPCOA) and the Air Resources Board (ARB), was issued in September 1990 to assist landfills statewide to control emissions (ARB, 1990j). The ARB is participating in the development of federal standards for POTWs. Although vinyl chloride is not produced in California, several thousand tons are used each year by two facilities producing polyvinyl chloride. However, a statewide control measure is generally not promulgated to address emissions primarily from two facilities.

The primary stationary sources that have reported emissions of vinyl chloride are sanitary services, airports/flying fields and services, and combination utility services (ARB, 1997b).

B. Emissions

The total emissions of vinyl chloride from stationary sources in California are estimated to be at least 18,000 pounds per year, based on data reported under the Air Toxics "Hot Spots" Program (AB 2588) (ARB, 1997b).

C. Natural Occurrence

Vinyl chloride monomer does not naturally occur in the environment (HSDB, 1995).

AMBIENT CONCENTRATIONS

Vinyl chloride has never been detected in samples collected by the statewide Air Resources Board (ARB) toxics monitoring network. Detectable levels of vinyl chloride in California are limited to locations near identified emission sources such as landfills. When vinyl chloride was formally identified as a toxic air contaminant, the ARB estimated by computer modeling,

population-weighted annual average vinyl chloride concentration for people living near a landfill

ranging from 1.56 to 23.4 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) or 0.6 to 9 parts per billion (ppb), and at another landfill from 1.56 to 26.0 $\mu\text{g}/\text{m}^3$ or 2 to 10 ppb (ARB, 1990d).

The United States Environmental Protection Agency (U.S. EPA) has reported concentrations of vinyl chloride from 13 study areas during 1989 to 1991. Overall range of concentrations from these areas were from 0.08 to 202 $\mu\text{g}/\text{m}^3$ or 0.03 to 77.69 ppb with an overall mean concentration of 0.96 $\mu\text{g}/\text{m}^3$ or 0.37 ppb (U.S. EPA, 1993a).

INDOOR SOURCES AND CONCENTRATIONS

With the exception of some homes located near landfills, indoor concentrations of vinyl chloride are not expected to be substantially greater than outdoor concentrations. Few indoor sources of vinyl chloride have been identified and most studies that have monitored for indoor concentrations of vinyl chloride fail to detect it. However, landfills have been identified as a source of emissions that contributes to elevated indoor levels of vinyl chloride in nearby residences. Grab samples from some houses located near landfills have shown vinyl chloride at concentrations up to 100 ppb. Other indoor sources include polyvinyl chloride products containing residues of vinyl chloride and cigarette smoke. For most homes, these sources are not expected to result in substantially elevated indoor levels of vinyl chloride (ARB, 1990d).

Homes that are located downwind from landfills can receive vinyl chloride through direct outdoor air influx into indoor environments. In addition, landfill gases containing vinyl chloride can migrate underground and enter homes through substructures. The rate of accumulation of vinyl chloride indoors depends on several factors including soil permeability, source strength, air exchange rate, and structure of the home. Higher indoor concentrations may occur because vinyl chloride is more rapidly destroyed in outdoor air than indoor air (ARB, 1990d).

During June of 1990, 125 households in Woodland, California were monitored for a variety of toxic air contaminants. Vinyl chloride was not found above the quantifiable limit of 1.2 $\mu\text{g}/\text{m}^3$ or 0.47 ppb either outdoors or indoors. These results suggest that few homes have measurable levels of vinyl chloride (Sheldon et al., 1992).

For another study conducted in California, fixed-site monitoring stations were installed to monitor indoor and outdoor air concentrations of vinyl chloride. Based on the analysis of 32 indoor samples taken in 8 homes during the summer season for 2 twelve-hour sampling periods (daytime and nighttime), concentrations of vinyl chloride were all below the limit of detection. The samples were analyzed by two analytical methods with limits of detection ranging from about 0.55 to 148 $\mu\text{g}/\text{m}^3$ or 0.2 to 58 ppb (Pellizzari et al., 1989).

A similar study was conducted in Baltimore where indoor air concentrations of vinyl chloride in about 160 homes were monitored by fixed-site sampling stations. Based on partially analyzed results, vinyl chloride was not detected in indoor environments. The limit of detection was reported to range from 26 to 40 $\mu\text{g}/\text{m}^3$ or 10.2 to 15.7 ppb (Pellizzari et al., 1987a).

ATMOSPHERIC PERSISTENCE

The dominant tropospheric loss process for vinyl chloride is by gas-phase reaction with the hydroxyl radical. The calculated half-life and lifetime are estimated to be 1.4 days and 2.1 days, respectively. The reaction forms formyl chloride and formaldehyde (Atkinson, 1995).

AB 2588 RISK ASSESSMENT INFORMATION

The Office of Environmental Health Hazard Assessment reviews risk assessments submitted under the Air Toxics “Hot Spots” Program (AB 2588). Of the risk assessments reviewed as of April 1996, vinyl chloride was the major contributor to the overall cancer risk in 7 of the approximately 550 risk assessments reporting a total cancer risk equal to or greater than 1 in 1 million and contributed to the total cancer risk in 28 of these risk assessments. Vinyl chloride also was the major contributor to the overall cancer risk in 4 of the approximately 130 risk assessments reporting a total cancer risk equal to or greater than 10 in 1 million, and contributed to the total cancer risk in 10 of these risk assessments (OEHHA, 1996a).

For non-cancer health effects, vinyl chloride contributed to the total hazard index in 3 of the approximately 89 risk assessments reporting a total chronic hazard index greater than 1 (OEHHA, 1996b).

HEALTH EFFECTS

The probable route of human exposure to vinyl chloride is inhalation.

Non-Cancer: Acute inhalation exposure to vinyl chloride has led to narcosis, respiratory irregularity, convulsions, cyanosis, and death. Less severe acute exposures have resulted in dizziness and headaches (U.S. EPA, 1994a). Vinyl chloride may sensitize the myocardium to the arrhythmogenic effects of epinephrine (Olson, 1994). Chronic inhalation exposure of workers to vinyl chloride has induced acro-osteolysis (degeneration of the terminal phalanges), vasospasm of the hands, dermatitis, circulatory and central nervous system alterations, thrombocytopenia, splenomegaly, and changes in liver function (ARB, 1990d).

A chronic non-cancer Reference Exposure Level (REL) of $26 \mu\text{g}/\text{m}^3$ is listed in the California Air Pollution Control Officers Association (CAPCOA) Revised 1992 Risk Assessment Guidelines for vinyl chloride. The toxicological endpoints considered for chronic toxicity are the gastrointestinal, liver and reproductive systems (CAPCOA, 1993). The U.S. EPA has not established either a Reference Concentration (RfC) or an oral Reference Dose (RfD) for vinyl chloride (U.S. EPA, 1994a).

Human studies have provided limited evidence of adverse effects on fetal development and male reproduction. According to the U.S. EPA, studies using rats have found decreased fetal weight and increased malformations at inhalation concentrations of vinyl chloride which also cause maternal toxicity. There are also reports of testicular damage after inhalation exposure in rats (ATSDR, 1993c; U.S. EPA, 1994a).

Cancer: In humans, epidemiological studies of occupationally exposed workers have linked vinyl chloride exposure to development of a rare cancer, liver angiosarcoma, and have suggested a relationship between exposure and lung and brain cancers. Chronic inhalation and oral exposures of rats, mice, and hamsters to vinyl chloride have been associated with an increased incidence of cancers at several sites including the liver, lung, mammary gland, and the nervous system (ARB, 1990d).

The U.S. EPA has classified vinyl chloride in Group A: Human carcinogen. The cancer potency number is under review by the U.S. EPA. However, the U.S. EPA previously calculated an inhalation unit risk estimate of 8.4×10^{-5} (microgram per cubic meter)⁻¹ and an oral unit risk of 5.4×10^{-5} micrograms per liter (U.S. EPA, 1994a). The International Agency for Research on Cancer has classified vinyl chloride in Group 1: Human carcinogen based on sufficient evidence of carcinogenicity in both humans and animals (IARC, 1987a).

The State of California has determined under Proposition 65 and AB 1807 that vinyl chloride is a carcinogen (CCR, 1996; ARB, 1990d). The inhalation potency factor that has been used as a basis for regulatory action in California is 7.8×10^{-5} (microgram per cubic meter)⁻¹ (OEHHA, 1994). In other words, the potential excess cancer risk for a person exposed over a lifetime to $1 \mu\text{g}/\text{m}^3$ of vinyl chloride is estimated to be no greater than 78 in 1 million. The oral potency factor that has been used as a basis for regulatory action in California is 2.7×10^{-1} (milligram per kilogram per day)⁻¹ (OEHHA, 1994).

